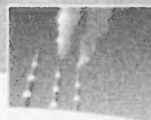
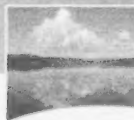




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# Data Sources and Methods for the Air Health Indicator

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# 1 Introduction

The Air Health Indicator (AHI) (<http://ec.gc.ca/indicateurs-indicateurs/default.asp?lang=en&n=CB7B92BA-1>) is part of the Canadian Environmental Sustainability Indicators (CESI) program (<http://ec.gc.ca/indicateurs-indicateurs/default.asp?lang=En&n=47F48106-1>), which provides data and information to track Canada's performance on key environmental sustainability issues. This indicator is also used to measure progress towards the goals and targets of the Federal Sustainable Development Strategy (<http://www.ec.gc.ca/dd-sd/default.asp?lang=En&n=CD30F295-1>).

## 2 Description and rationale of the Air Health Indicator

### 2.1 Description

The AHI tracks the percentage of all cardiopulmonary mortality risks (deaths from heart- and lung-related diseases) that can be attributed to exposure to two important outdoor air pollutants: ground-level ozone ( $O_3$ ) and fine particulate matter ( $PM_{2.5}$ ).

### 2.2 Rationale

Canadians are regularly exposed to air pollution from outdoor sources such as transportation and industrial activities. This exposure can lead to the onset or worsening of breathing difficulty, the development of chronic lung disease or heart attacks and strokes. These health effects contribute to lost productivity, increased doctors' and emergency room visits and hospital admissions, and mortality. The AHI has been developed as a tool to monitor the impacts of outdoor air pollution exposure over time on the health of Canadians.

### 2.3 Recent changes

More recent mortality data for the years 2005 to 2007 were obtained for this version of the AHI. The methodology essentially remained the same except for the application of the Bayesian hierarchical model where a level was added for this new AHI. Previously the statistical model considers risk from cities and then from the national level. The current version considers risks at the city, then regional and at the national level. Minor changes also came from the use of newer and updated measurements ( $PM_{2.5}$  and  $O_3$ ) and the introduction of measurements from new  $PM_{2.5}$  monitoring equipment.

## 3 Data

Canadian communities for which the ground-level  $O_3$  and  $PM_{2.5}$  concentrations were used for the National Air Quality Indicators (<http://ec.gc.ca/indicateurs-indicateurs/default.asp?lang=en&n=7DCC2250-1>) of CESI were considered. The AHI is based on the criteria of having a reasonably complete time series of pollution and weather measurements, and enough daily mortality data.

For each community there were three types of data used for the AHI: daily numbers of cause-specific deaths, air pollution concentrations, and potential confounders to the mortality-air pollution association.

### 3.1 Data source

#### 3.1.1 Daily numbers of cause-specific deaths

The daily numbers of cause-specific deaths (non-accidental mortality data) were obtained from the national mortality database (Vital Statistics Database-Deaths [[http://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&SDDS=3233&Item\\_Id=144261&lang=en](http://www23.statcan.gc.ca/imdb/p2SV.pl?Function=getSurvey&SDDS=3233&Item_Id=144261&lang=en)]) maintained by Statistics Canada. Based on the International Classification of Diseases (ICD), the mortality data included only deaths from internal causes (ICD-9 code < 800 for up to year 1999 and ICD-10 code A00-R99 for years 2000 onwards), excluding external causes such as injuries. Regarding cause-specific deaths, in particular, we were interested in cardiopulmonary mortality related to the circulatory or respiratory system. For this specification, our mortality data were categorized into a cardiopulmonary group (ICD-9 code between 390 and 520 and ICD-10 code between I00-I99 and J00-J99). The cardiopulmonary mortality data were extracted for a specified census division only where the census division of residence was the same as the census division of death occurrence.

#### 3.1.2 Air pollution concentrations

The daily  $O_3$  and  $PM_{2.5}$  concentration data were obtained from the National Air Pollution Surveillance (NAPS) Network (<http://www.ec.gc.ca/rnspar-naps/Default.asp?lang=En&n=5C0D33CF-1>) operated by Environment Canada. Established in 1969, NAPS provides accurate and long-term air quality data of a uniform standard across Canada to monitor the quality of ambient (outdoor) air in populated regions by specific procedures for the selection and positioning of monitoring stations. For each NAPS monitoring station, the daily average concentration for a certain day was calculated only if at least 75% of 24 hourly concentrations for that day (i.e. at least 18 hourly concentrations) were available. Otherwise, it was recorded as missing. For each census division, the daily average concentration was averaged over monitoring stations if there were two or more stations located in that census division. The metrics used for the concentrations were the daily 8-hours maximum (April to October) for  $O_3$  and the daily mean (April to October) for  $PM_{2.5}$ .

#### 3.1.3 Potential confounders to the mortality-air pollution association

As for potential confounding variables to the exposure-mortality association, three factors were considered: time; temperature; and indicators for days of the week. Calendar time is included to control both temporal and seasonal variations. Daily temperature controls for the short-term effect of weather on daily mortality; and day of the week accounts for mortality that varies by day of the week. Specifically, to account for the weather effect, daily mean temperature data were obtained from the National Climate Data and Information Archive ([http://climate.weatheroffice.gc.ca/Welcome\\_e.html](http://climate.weatheroffice.gc.ca/Welcome_e.html)) of Environment Canada. As for lifestyle factors such as smoking or cholesterol in the community, they do not vary meaningfully from day to day and thus can be ignored as confounders.

### 3.2 Spatial coverage

Twenty Canadian communities<sup>1</sup> were selected for  $O_3$ . Eighteen communities<sup>2</sup> were selected for  $PM_{2.5}$ . Each community's geographic boundaries were defined by the census division associated

<sup>1</sup> Saint John, Québec, Montréal, Ottawa, York, Toronto, Peel, Halton, Hamilton, Niagara Falls, Waterloo, Windsor, Sarnia, Sault Ste. Marie, Winnipeg, Regina, Saskatoon, Calgary, Edmonton, and Vancouver.

<sup>2</sup> Saint John, Québec, Montréal, Ottawa, Toronto, Peel, Halton, Hamilton, Niagara Falls, London, Windsor, Sarnia, Waterloo, Winnipeg, Regina, Calgary, Edmonton, and Vancouver.

with the city.

### 3.3 Temporal coverage

Yearly data for the years 1990 to 2010 were used for  $O_3$  and yearly data for the years 2001 to 2010 were used for  $PM_{2.5}$ .

### 3.4 Data completeness

At the time of the modeling of the AHI, only the 1990 to 2007 mortality data were sufficiently complete and available in the correct format. The indicators values reported for years 2008 to 2010 should be considered as preliminary, as they are approximated using the averages of annual national risk estimates from the previous periods (1990 to 2007 for  $O_3$  and 2001 to 2007 for  $PM_{2.5}$ ). A reasonable assumption was made that the consistency observed in these estimates continued. The latest year used for the air pollution concentration is 2010.

### 3.5 Data timeliness

Due to the complexity of mortality data collection, the AHI is few years behind the other data (air pollution concentrations).

## 4 Methods

The AHI is based on two temporal functions: annual  $O_3$  and  $PM_{2.5}$  concentrations and annual mortality risks at the national level. The annual air pollutant concentrations were obtained from the NAPS Network (<http://www.ec.gc.ca/rnsps-naps/Default.asp?lang=En&n=5C0D33CF-1>).

To identify the cardiopulmonary mortality-air pollution association and establish annual mortality risks, a Bayesian 2-level hierarchical model was applied. First, the city-specific risk for each city (census division) was estimated. Annual city-specific risks of cardiopulmonary mortality due to  $O_3$  or  $PM_{2.5}$  were estimated by a generalized Poisson model for each census division. To handle potential overdispersion with daily mortality counts and to account for confounders, a generalized additive over-dispersed Poisson regression model was applied. Second, the national risks for each year were estimated by pooling over the city-specific risks within region and between regions simultaneously. Finally, testing was done of the resulting data for annual national attributable risks (due to  $O_3$  or  $PM_{2.5}$ ) to determine whether a time trend could be detected.

The annual cardiopulmonary mortality risk estimates showed no upward or downward trend. It was therefore possible to assert that the annual mortality risk was constant over the period analysed. The average of 18 annual national risk estimates for  $O_3$  (0.011 per 10 parts per billion (ppb) for 1990 to 2007) and the average of eight annual national risk estimates for  $PM_{2.5}$  (0.025 per 10 ppb for 2001 to 2007) were used. It was assumed that this constancy persisted and each of these averages was projected for the years 2008 to 2010, due to the lack of mortality data to develop risks estimates for these years.

The overall annual risk was derived from the product of annual air pollutant concentrations and the average of annual mortality risk estimates of  $O_3$  and  $PM_{2.5}$ . Finally, a time-trend analysis was also performed on the AHI using the Sen's test, a non-parametric linear trend test. A trend was detected at the 95% confidence interval for the  $O_3$  AHI but not for  $PM_{2.5}$ .



## 5 Caveats and limitations

The AHI is an indicator in development. It is concentrating on the mortality risk aspect from cardiopulmonary diseases as a whole for O<sub>3</sub> and PM<sub>2.5</sub> in communities where we had the best available data. Up to now the AHI work does not include assessments of the potential reasons behind changes in mortality attributable to air pollutant exposure.

## 6 References and further reading

### 6.1 References

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